Field trials of a new sub-acute rodenticide flupropadine, against wild Norway rats (*Rattus norvegicus*)

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SUMMARY

Fifteen experimental treatments with rodenticidal baits containing 0·1, 0·15 or 0·2 % flupropadine were conducted on farmsteads against *Rattus norvegicus* infestations. Eight treatments were completely successful and the others gave kills ranging from 36 to 72 % in 28 days. Treatments with 0·1 and 0·15 % flupropadine were less successful against large infestations than against small ones. Flupropadine was most successful at 0·2 % but still gave incomplete kills on farms where abundant alternative food was available. The compound was more effective than acute poisons in achieving complete control of Norway rat infestations, but was less reliable in doing so than anticoagulants. On the other hand, many flupropadine treatments gave quicker control and used smaller quantities of bait than anticoagulant treatments.

INTRODUCTION

Anticoagulant rodenticides have been used for the control of Norway rats (*Rattus norvegicus*) for about 30 years. However, their predominant position was threatened by the discovery of warfarin resistance in various parts of the United Kingdom (Boyle, 1960; Greaves & Rennison, 1973). This led to the development of the potent second-generation anticoagulants that are effective against warfarin-resistant rodents (Hadler & Shadbolt, 1975; Grand, 1976). In spite of this success, interest was maintained in rodenticides with different modes of action (e.g. Rennison, 1974*a,b*; 1976). Recently, this work assumed greater importance with the discovery, in Hampshire, of resistance to difenacoum, one of the second-generation anticoagulants, and with the suggestion that further losses of efficacy of these compounds might be expected (Greaves, Shepherd & Quy, 1982).

During the routine laboratory screening of new compounds for rodenticidal activity, one, flupropadine (1-(3,5-bistrifluromethyl phenyl)-3(4-tertbutyl piperidino)-prop-1-yne, as the hydrochloride), showed particular promise (unpublished data). Norway rats, offered bait poisoned with flupropadine in free-feeding tests, typically ate well for 2 days but little thereafter, and died between 4 and 9 days after the initial presentation of the poison. These characteristics do not fit either the acute or chronic categories that are often used to classify rodenticidal compounds; the action of flupropadine therefore is best described as sub-acute, a term also applied to calciferol (Rennison, 1974*a*).

The field trials described in this paper were conducted to assess the efficacy of flupropadine for the control of Norway rat populations living in and around farm buildings in areas of Shropshire and Powys where trials of a number of new rodenticides have been conducted in the past and where, on average, half the rats on each farm are resistant to warfarin.

METHODS

The farms used for the trials were selected so as to avoid both very lightly and very heavily infested premises (Rennison, 1974b). Laboratory tests had shown that the optimum concentration of flupropadine for field use might fall between 0·1 and 0·2%. Accordingly, three concentrations of the poison were tested (0·1, 0·15 and 0·2%) and farms were randomly allocated to be treated with one of the three concentrations until five had been treated with each.

For each treatment, the farmstead was surveyed to determine the extent of the rat infestation and, on a Thursday or Friday, clean wooden bait trays were put down at sites where rats were active. The numbers of trays laid (Figs. 1–3) thus provided a rough index of the size of each infestation. Baits were prepared by mixing cereal-based concentrates containing 2, 3 or 4% flupropadine, in the proportion 1 part of the concentrate to 19 parts of medium oatmeal. On the following Monday, 100 g of poisoned bait was placed in each bait tray and, thereafter, the farms were visited daily, Monday to Friday, to record the numbers of bait points visited by rats and to weigh and replenish the bait. Natural cover was used to protect the bait from the weather and from non-target animals. On some occasions the medium oatmeal bait base was replaced by soaked wheat where the dry bait was failing to attract rats that were consuming naturally available foods. A patch of fine sand or basic slag was put down beside each bait tray, and smoothed over at each visit after recording any rat tracks made since the previous day. Shepherd & Greaves (1984) discussed the assessment of tracking activity as a census technique for use in field trials of rodenticides. The treatments continued either until all signs of rat activity had ceased or for 4 weeks. On farms where incomplete control was achieved the level of kill was estimated from the relationship:

$$\% \text{ kill} = 1 - \frac{\text{mean proportion patches tracked days 23–25}}{\text{proportion patches tracked day 1}} \times 100.$$

RESULTS

Initially, flupropadine was well accepted in bait at all of the three different concentrations tested (Table 1; Figs. 1–3). The daily amount of bait eaten fell sharply during the first week of the treatments and, thereafter, only small numbers of takes were recorded.

The results of the 15 treatments were quite variable. Complete control was rapidly achieved on eight farms. Bait takes declined more quickly than tracking activity, probably because the poison first acts to depress appetite and only later exerts its lethal effect. On the other seven farms, tracking activity was recorded throughout the 28-day trial period. On several of these, after an initial decline,

Table 1. *The amounts of bait eaten during the* 28-*day period of field trials of different concentrations of flupropadine for the control of* R. norvegicus

(The figures represent the means of five trials of each concentration.)

Daily bait take

Day	0·1%		0·15%		0·2%	
	Weight (g)	%	Weight (g)	%	Weight (g)	%
1	1745	100	1550	100	1945	100
2	815	47	1140	74	835	43
3	235	13	480	31	320	16
4	150	9	240	15	310	16
5–7*	90	5	127	8	123	6
8	65	4	180	12	80	4
9	70	4	115	7	50	3
10	40	2	25	2	10	1
11	15	1	40	3	15	1
12–14*	10	1	43	3	17	1
15	40	2	0	0	0	0
16	116	7	10	1	30	2
17	105	6	5	1	60	3
18	95	5	5	1	30	2
19–21*	63	4	8	1	10	1
22	115	7	5	1	30	2
23	140	8	15	1	5	1
24	70	4	0	0	5	1
25	70	4	20	1	15	1
26–28*	27	2	0	0	0	0

* Mean daily bait take at weekends.

rat activity recovered somewhat and remained high, relative to the small quantities of poisoned bait eaten. This suggested that some animals had been sublethally poisoned at the beginning of the treatments, had recovered, and then did not feed further at the bait points.

With 0·1% flupropadine, complete control was achieved on two farms (1 and 2) within 9 days (Fig. 1), but the numbers of bait points set out and the weights of bait eaten on the first night indicated that the infestations were relatively small. On the remaining farms, two of which (3 and 4) were more heavily infested, bait takes fell steadily during the first 2 weeks of the treatments and thereafter fluctuated appreciably. This probably occurred on farms 3 and 4 because, during the third week, soaked wheat was substituted for medium oatmeal as the bait base, but there was no readily apparent reason for the fluctuations in bait uptake and tracking activity observed on farm 5. Kills estimated at 53, 57 and 72% were obtained on farms 3, 4 and 5 respectively.

The treatments on farms 6–8 in which 0·15% flupropadine was used were completely effective within 15 days (Fig. 2). Once again, two of these infestations were relatively small. On two more heavily infested farms, control was estimated at 48% (farm 9) and 66% (farm 10) when the treatments were terminated after 4 weeks.
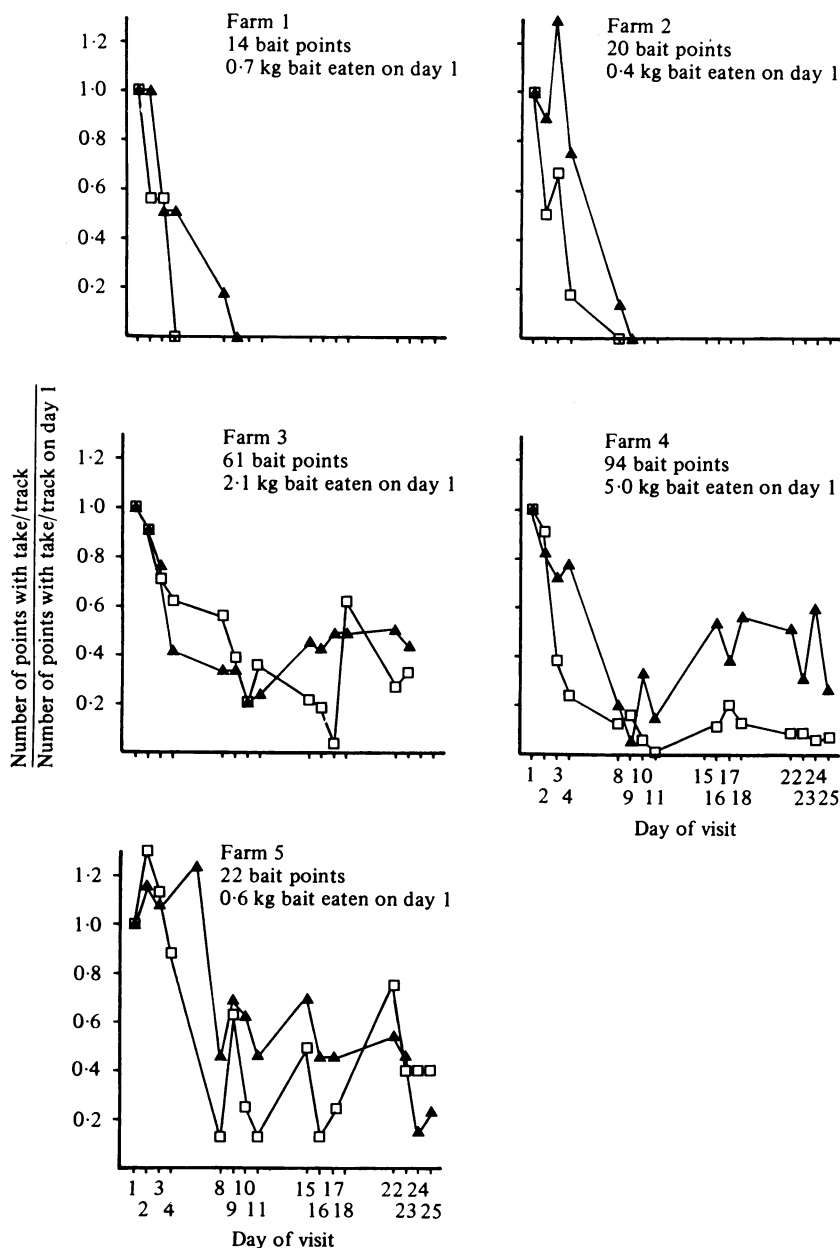
Fig. 1. The results of baiting with 0·1 % flupropadine on farms 1–5, demonstrated by the declines in the daily proportion of tracking patches with rat signs (▲) and the daily proportion of bait points with takes (□).

The 0·2 % flupropadine bait was also completely effective on three of the farms on which it was used (Fig. 3). Two relatively heavily infested farms (11 and 12) were cleared of rats within 15 days. On a third more lightly infested farm (13) activity was reduced to a low level in about the same period, and finally ceased on day 28. Incomplete control was achieved on two lightly infested farms, where 36 %
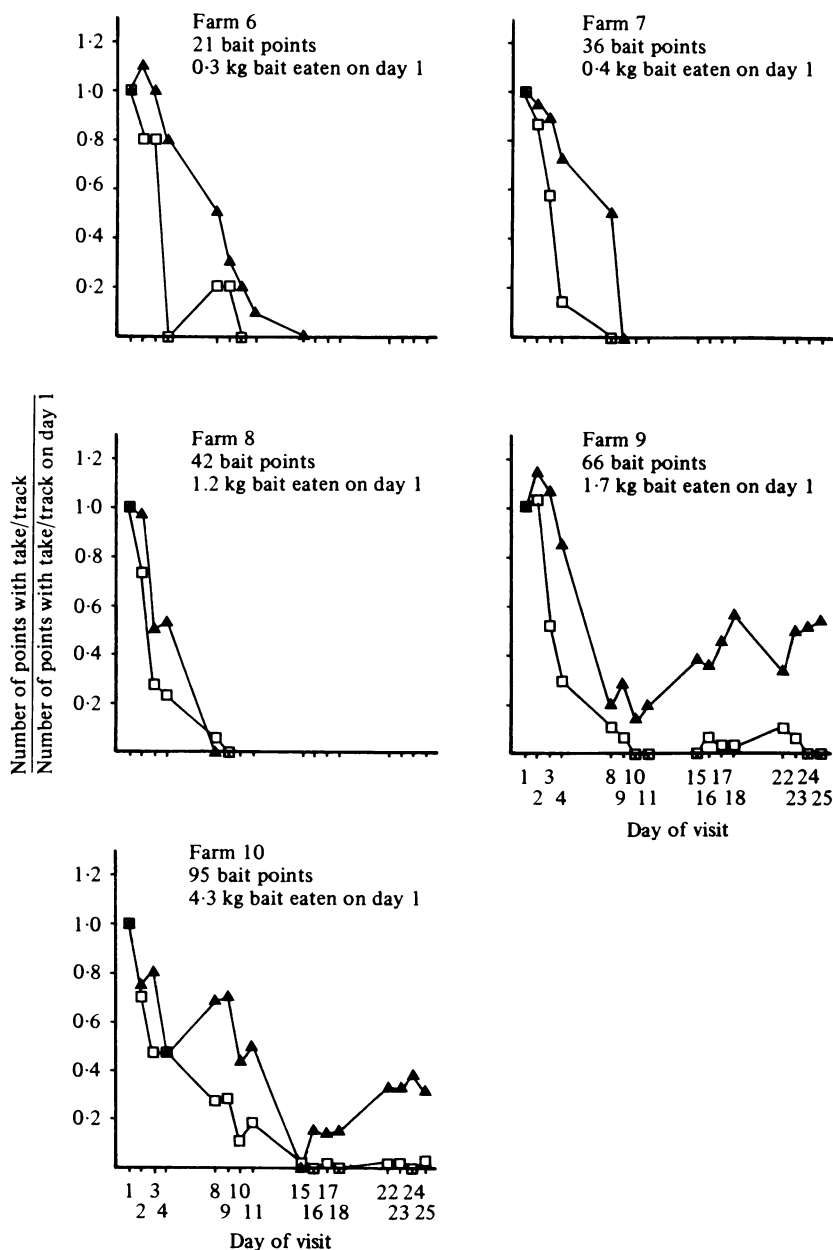
Fig. 2. The results of baiting with 0·15 % flupropadine on farms 6–10, demonstrated by the declines in the daily proportion of tracking patches with rat signs (▲) and the daily proportion of bait points with takes (□).

(farm 14) and 61 % (farm 15) reduction in tracking activity resulted from the treatments. The reasons for these failures cannot be determined with certainty but, on both farms, exceptionally large quantities of an attractive alternative food, meat, were readily available and may have caused poor bait acceptance.
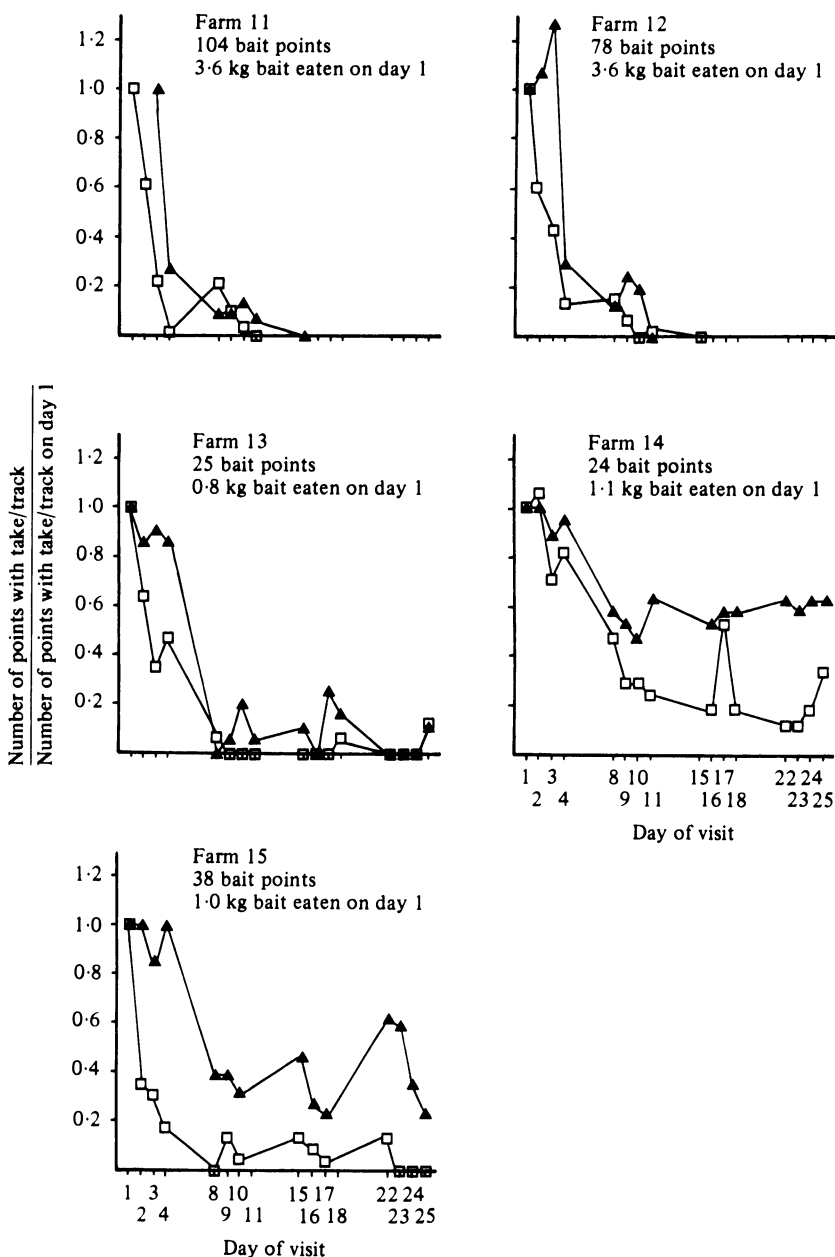
Fig. 3. The results of baiting with 0·2 % flupropadine on farms 11–15, demonstrated by the declines in the daily proportion of tracking patches with rat signs (▲) and the daily proportion of bait points with takes (☐).

DISCUSSION

Complete mortality occurred in laboratory tests when Norway rats were offered bait containing either 0·1 or 0·2 % flupropadine (unpublished data) but inconsistent results were obtained when these concentrations of the poison, and the intermediate one of 0·15 %, were tested in the field. Infestation size played a part in determining

treatment success when the lower concentrations (0·1 and 0·15 %) were employed; large infestations proving more difficult to control than smaller ones. At the higher concentration of 0·2 %, however, flupropadine successfully eradicated three *R. norvegicus* infestations, two of these being among the largest encountered. The failure of 0·2 % flupropadine to control two small populations can be attributed mainly to the abundance of attractive alternative food.

The rapid action of flupropadine is such that the time available for rats to ingest a lethal dose is much less than for anticoagulants, and is probably little more than 2 days. In flupropadine treatments, bait take decreased on the third day, on average, to 20 % of the first day's take (Table 1), as compared with a mean increase of 40 % during the equivalent period of three anticoagulant trials described by Drummond & Rennison (1973). A disadvantage of this rapid action is that animals which take only small quantities of bait during the first 2 days may be sublethally poisoned and, as a result, develop an aversion from it. This seems to have occurred in some trials of 0·1 and 0·15 % flupropadine (Fig. 1, farms 3, 4 and 5; Fig. 2, farms 9 and 10). In conducting flupropadine treatments, therefore, it is important to encourage rats to feed freely on the poisoned bait from the beginning. To do this, it is essential to avoid misjudgements in the initial bait placements that might lead to underbaiting, and desirable to reduce the availability of alternative food as far as possible. It seems unlikely that flupropadine will perform well in adverse conditions unless these steps are taken. Nevertheless, the trials showed the compound to be more effective than acute poisons in controlling Norway rats. For example, Rennison (1976) tested thallium sulphate, gophacide and zinc phosphide, after pre-baiting for 5 days, and eradicated the rats on only 2 of 30 farms treated. In comparison, rats were completely controlled on 8 out of 15 farms when flupropadine was used. Although flupropadine was less consistent than anticoagulants in eradicating rats (e.g. Rennison & Dubock, 1978; Richards, 1981), it often gave quicker and more economical results; all but one of the successful trials were completed within 14 days and used much less bait than is required in conventional anticoagulant treatments. In practice, few farmers continue poison-baiting treatments until complete control is achieved, and many users may prefer to accept the possibility, when using flupropadine, that some rats may survive, provided an acceptable level of kill is achieved quickly and economically.

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